We claim:

1. A method for controlling aberrant cell proliferation comprising

a) contacting a cell population comprising aberrantly proliferating cells with at least one Chk1 activator in am amount sufficient to substantially synchronize cell cycle arrest among said aberrantly proliferating cells at a target phase, and

b) upon achieving said substantial synchronization of cell cycle arrest among said aberrantly proliferating cells, contacting said cell population with a selective Chk1 inhibitor in an amount sufficient to substantially abrogate said cell cycle arrest.

2. The method of claim 1, wherein said Chk1 inhibitor is a specific Chk1 inhibitor.

3. The method of claim 1, wherein said cell population is contacted with a Chk1 activator for from about 30 minutes to about 96 hours, and subsequently contacted with a selective Chk1 inhibitor for from up to about 1 hour to up to about 72 hours.

4. The method of claim 3, wherein said cell population is contacted with a Chk1 activator for from about 30 minutes to about 48 hours.

5. The method of claim 1, wherein said Ck1 activator induces substantial synchronization of cell cycle arrest cells at the target phase G1.

6. The method of claim 1, wherein said Chk1 activator induces substantial synchronization of cell cycle arrest at the target phase S.

7. The method of claim 1, wherein said Chk1 activator induces substantial synchronization of cell cycle arrest at the target phase G2.

8. The method of claim 1, wherein said cell population is *ex vivo*.

9. The method of claim 1, wherein said cell population is *in vivo*.

10. The method of claim 9, wherein said cell population is in a human.

11. The method of claim 1, wherein said Chk1 activator comprises a chemotherapeutic agent.

12. The method of claim 1, wherein said Chk1 activator is an alkylating agent.

13. The method of claim 12, wherein said alkylating agent is a nitrogen mustard.

14. The method of claim 13, wherein said nitrogen mustard is mechlorethamine, cyclophosphamide, ifosfamide, melphalan, or chlorambucil.

15. The method of claim 1, wherein said Chk1 activator is a nitrosourea.

16. The method of claim 15, wherein said nitrosourea is carmustine (BCNU), lomustine (CCNU), or semustine (methyl-CCNU).

17. The method of claim 1, wherein said Chk1 activator is an ethylenimine or a methyl-melamine.

18. The method of claim 17, wherein said ethylenimine or said methyl-melanine is triethylenemelamine (TEM), triethylene thiophosphoramide (thiotepa), or hexamethylmelamine (HMM, altretamine).

19. The method of claim 1, wherein said Chk1 activator is an alkyl sulfonate.

20. The method of claim 19, wherein said alkyl sulfonate is busulfan.

21. The method of claim 1, wherein said Chk1 activator is a triazine.

22. The method of claim 21, wherein said triazine is dacarbazine (DTIC).

23. The method of claim 1, wherein said Chk1 activator is an antimetabolite.

24. The method of claim 23, wherein said antimetabolite is a folic acid analog.

25. The method of claim 24, wherein said folic acid analog is methotrexate, trimetrexate, or pemetrexed (multi-targeted antifolate).

26. The method of claim 23, wherein said antimetabolite is a pyrimidine analog.

27. The method of claim 26, wherein said pyrimidine analog is 5-fluorouracil (5-FU), fluorodeoxyuridine, gemcitabine, cytosine arabinoside (AraC, cytarabine), 5-azacytidine, or 2,2'-difluorodeoxycytidine.

28. The method of claim 23, wherein said antimetabolite is a purine analog.

29. The method of claim 28, wherein said purine analog is 6-mercaptopurine, 6-thioguanine, azathioprine, 2'-deoxycoformycin (pentostatin), erythrohydroxynonyladenine (EHNA), a fludarabine salt, or 2-chlorodeoxyadenosine (cladribine, 2-CdA).

30. The method of claim 23, wherein said antimetabolite is a type I topoisomerase inhibitor.

31. The method of claim 30, wherein said type I topoisomerase inhibitor is camptothecin (CPT), topotecan, or irinotecan.

32. The method of claim 1, wherein said Chk1 activator is derived from a natural product.

33. The method of claim 32, wherein said natural product is a epipodophylotoxin.

34. The method of claim 33, wherein said epipodophylotoxin is etoposide or teniposide.

35. The method of claim 32, wherein said natural product is a vinca alkaloid.

36. The method of claim 35, wherein said vinca alkaloid is vinblastine, vincristine, or vinorelbine.

37. The method of claim 1, wherein said Chk1 activator is an antibiotic.

38. The method of claim 37, wherein said antibiotic is actinomycin D, doxorubicin, or bleomycin.

39. The method of claim 1, wherein said Chk1 activator is a radiosensitizer.

40. The method of claim 39, wherein said radiosensitizer is 5-bromodeozyuridine, 5-iododeoxyuridine, or bromodeoxycytidine.

41. The method of claim 1, wherein said Chk1 activator is a platinum coordination complex.

42. The method of claim 41, wherein said platinum coordination complex is a cisplatin, carboplatin, or oxaliplatin.

43. The method of claim 1, wherein said Chk1 activator is hydroxyurea.

44. The method of claim 1, wherein said Chk1 activator is a methylhydrazine derivative.

45. The method of claim 44, wherein said methylhydrazine derivative is N-methylhydrazine (MIH) or procarbazine.

46. The method of claim 1, wherein said Chk1 activator comprises radiation.

47. The method of claim 46, wherein said radiation is x-ray radiation or ultraviolet radiation.

48. The method of claim 47, wherein said radiation is administered in conjunction with a radiosensitizer and/or a photosensitizer.

49. The method of claim 1, further comprising the administration of at least one chemotherapeutic agent or at least one radiotherapeutic agent that does not activate Chk1.

50. The method of claim 1, further comprising the administration of at least one side effect reducing agent.

51. The method of claim 1, wherein said cell population is contacted with a Chk1 inhibitor after a time sufficient to allow said Chk1 activator to induce a maximum degree of synchronization in said cell population of cell cycle arrest and a minimum number of cells in mitosis.

52. The method of claim 1, wherein the substantially synchronized cell cycle arrest achieved by contacting said cell population with said Chk1 activator comprises at least about a 50% increase in the number of aberrantly proliferating cells in the target phase of said Chk1 activator in comparison to the number of aberrantly proliferating cells in the target phase prior to contact with said Chk1 activator.

53. The method of claim 52, wherein said increase is at least about 100%.

54. The method of claim 53, wherein said increase is at least about 200%.

55. The method of claim 54, wherein said increase is at least about 300%.

56. The method of claim 55, wherein said increase is at least about 400%.

57. The method of claim 1, wherein said cell population is contacted with said Chk1 activator for at least one doubling period typical of aberrantly proliferating cells in said cell population.

58. The method of claim 1, wherein said cell population is contacted with said Chk1 activator for at least two doubling periods typical of aberrantly proliferating cells in said cell population.

59. The method of claim 1, further comprising determining the presence or absence of substantial synchronization of cell cycle arrest in a biological sample.

60. The method of claim 59 wherein the biological sample is a fluid sample or a tissue sample.

61. The method of claim 1, wherein said Chk1 inhibitor is administered over a plurality of doses.

62. The method of claim 25, wherein said doses comprise a frequency of (q4d x 4), (q3d x 4), (qd x 5), (qwk3), or (5/2/5).

63. The method of claim 1, wherein said aberrantly proliferating cells are cancerous.

64. The method of claim 63, wherein said cancerous cells comprise cells from myxoid and round cell carcinomas, locally advanced tumors, metastatic cancer, Ewing's sarcoma, cancer metastases, lymphatic metastases, squamous cell carcinomas, esophageal squamous cell carcinomas, oral carcinomas, multiple myelomas, acute lymphocytic leukemias, acute non-lymphocytic leukemias, chronic lymphocytic leukemias, chronic myelocytic leukemias, hairy cell leukemias, effusion lymphomas (body cavity based lymphomas), thymic lymphoma lung cancers, small cell carcinomas of the lung, cutaneous T cell lymphomas, Hodgkin's lymphomas, non-Hodgkin's lymphomas, cancers of the adrenal cortex, ACTH-producing tumors, non-small cell lung cancers, breast cancers, small cell carcinomas, ductal carcinomas, stomach cancers, colon cancers, colorectal cancers, polyps associated with colorectal neoplasias, pancreatic cancers, liver cancers, bladder cancers, primary superficial bladder tumors, invasive transitional cell carcinomas of the bladder, muscle-invasive bladder cancers, prostate cancers, ovarian carcinomas, primary peritoneal epithelial neoplasms, cervical carcinomas, uterine endometrial cancers, vaginal cancers, cancers of the vulva, uterine cancers and solid tumors in the ovarian follicle, testicular cancers, penile cancers, renal cell carcinomas, intrinsic brain tumors, neuroblastomas, astrocytic brain tumors, gliomas, metastatic tumor cell invasions in the central nervous system, osteomas and osteosarcomas, malignant melanomas, tumor progressions of human skin keratinocytes, squamous cell cancers, thyroid cancers, retinoblastomas, neuroblastomas, peritoneal effusions, malignant pleural effusions, mesotheliomas, Wilms's tumors, gall bladder cancers, trophoblastic neoplasms, hemangiopericytomas, Kaposi's sarcomas or other cancers treatable with chemotherapy agents or inhibitors of cell cycle checkpoint proteins.

65. The method of claim 1, wherein said aberrantly proliferating cells are non-cancerous.

66. The method of claim 63, wherein said non-cancerous cells comprise cells originating from atherosclerosis, restenosis, vasculitis, nephritis, retinopathy, renal disease, proliferative skin disorders, psoriasis, keloid scarring, actinic keratosis, Stevens-Johnson Syndrome, rheumatoid arthritis, systemic-onset juvenile chronic

arthritis, osteoporosis, systemic lupus erythematosus, hyperproliferative diseases of the eye including epithelial down growth, proliferative vitreoretinopathy (PVR), hemangio-proliferative diseases, ichthyosis, or papillomas.

67. Use of a composition comprising at least one Chk1 inhibitor in the manufacture of a medicament for the inhibition of aberrant cell proliferation.

68. An article of manufacture comprising a Chk1 inhibitor and a label indicating a method according to claim 1.